Radiology Case Reports 20 (2025) 1182–1188

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

# Magnetic resonance imaging in diagnosing spinal cord tuberculoma: A case series and literature review ☆,☆☆

*Shimalis Fayisa, MD*[a,*], *Sherief Ghozy, MD*[b], *Armin Zarrinttan, MD*[b], *Cem Bilgin, MD*[b], *David F. Kallmes, MD*[b]

[a] *Department of Radiology, Care Land General Hospital, Addis Ababa, Ethiopia*
[b] *Neuro-Vascular Research Lab, Department of Radiology, Mayo Clinic, Rochester, MN, USA*

ARTICLE INFO

*Article history:*
Received 17 October 2024
Revised 24 October 2024
Accepted 30 October 2024

*Keywords:*
Spinal cord tuberculoma
MRI
T2 hypointensity
Target sign

ABSTRACT

Spinal cord tuberculoma is a clinically significant form of extra-pulmonary tuberculosis, despite its rarity. It leads to considerable neurological deficits and morbidity. Accurate diagnosis and early intervention depend on radiologic imaging, typically MRI, which reveals T2 hypointensity with rim enhancement, forming a "target sign" characteristic of caseous stage tuberculoma. In this article, we present 3 cases of spinal cord tuberculoma without adjacent vertebral involvement; 2 cases affect the lower thoracic region, while one involves the cervical cord. All patients exhibited some degree of body weakness, which improved following the initiation of anti-tuberculosis treatment. Also, we discuss the role of MRI in diagnosing spinal cord tuberculoma, highlighting its characteristic findings, and review recent literature on the topic.

© 2024 The Authors. Published by Elsevier Inc. on behalf of University of Washington.
This is an open access article under the CC BY-NC-ND license
(http://creativecommons.org/licenses/by-nc-nd/4.0/)

## Introduction

Tuberculosis (TB) is one of the most ancient known diseases affecting humanity, with evidence of its presence in ancient Egyptian and Peruvian mummies dating back to 9000 BC [1].

The global burden of tuberculosis is rising, including development of drug-resistant strains and severe forms of the disease. This increase is likely linked to the growing global HIV burden and the rising use of chemotherapy and steroids, which can impair immunity. According to the WHO's 2022 Global Tuberculosis Report, an estimated 10.6 million people fell ill with TB in 2021, up from 10.1 million in 2020.

*Abbreviations:* AFB, Acid-Fast Bacilli; CNS TB, central nervous system tuberculosis; FNAC, fine-needle aspiration cytology; TB, tuberculosis; CBC, complete blood count; ESR, erythrocyte sedimentation rate.

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

☆☆ Acknowledgments: The authors acknowledge the assistance of Sonia Watson, PhD, in editing and formatting of the manuscript.

* Corresponding author.
E-mail address: shimalis.tadesse379@gmail.com (S. Fayisa).

https://doi.org/10.1016/j.radcr.2024.10.156

1930-0433/© 2024 The Authors. Published by Elsevier Inc. on behalf of University of Washington. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/)

Additionally, 1.6 million people died from TB in 2021 compared to 1.5 million in 2020 [2].

Tuberculosis is relatively common in certain regions of the world and can potentially affect almost every part of the human body. Central nervous system tuberculosis (CNS TB) is rare however, affecting only 0.5%-2.0% of individuals with systemic TB, with 2 out of 100,000 involving the spinal cord [3].

CNS TB can manifest in 3 distinct forms: tuberculous meningitis, intracranial tuberculoma, or spinal TB. Spinal TB can present with a variety of lesions, including tuberculous spondylitis, arachnoiditis, meningitis, or intramedullary lesions. Among these, intramedullary tuberculosis (tuberculoma) is the least prevalent, but imposes a significant clinical impact and accounts for approximately 8% of all cases of spinal TB [1].

Spinal cord TB is a significant extrapulmonary form of the disease, primarily affecting individuals aged 16-30. Its prevalence has increased, likely due to rising HIV/AIDS cases. The advent of CT and MRI since 1987 has enhanced diagnostic capabilities, helping to prevent permanent neurological deficits [1,4].

MRI is the preferred modality for evaluating potential tuberculomas. Although the imaging appearance can be classical and strongly indicative of a diagnosis—characterized by central low T2 signals, surrounding enhancement, and edema— it is important to recognize that these features may vary depending on the stage of the disease [1].

In this article, we summarize the MRI findings from 3 cases of spinal cord tuberculoma confirmed through clinical evaluation, laboratory tests, and MRI imaging. We also review the 4 stages of tuberculoma and their characteristic MRI findings with relevant literature to update clinicians, aiding in the early diagnosis and management of this rare and challenging condition to minimize the risk of permanent neurological sequelae.

### *Case 1*

A 25-year-old male schoolteacher presented with a 2-day history of acute urinary retention, along with numbness and bilateral weakness in his lower limbs that had developed over the past 10 days. He also reported a persistent productive cough for the past 2 months, as well as weight loss and decreased appetite. The patient has a history of contact with a known TB patient—his brother—2 years ago. A complete blood count (CBC) showed normal results, but the erythrocyte sedimentation rate (ESR) was elevated. Sputum tests for Acid Fast Bacilli (AFB) were conducted and returned positive on 2 occasions, confirming the presence of Mycobacterium tuberculosis.

Multiplanar spinal MRI (thoracolumbar) without and with contrast was performed, given that the patient had already exhibited localized signs of lower extremity weakness. The MRI revealed a small intradural, extra medullary lesion at the L2 level of the lumbar spine, measuring approximately 12 × 16 mm. The lesion was isointense on T1-weighted images and isointense to hypointense on T2-weighted and T2 fat-suppressed STIR images, with a small focus of central hyperintensity on long TR sequences. The lesion demonstrated homogeneous enhancement with a small central nonenhancing focus on postcontrast imaging (Fig. 1).

Based on the laboratory findings (positive sputum AFB) and the characteristic MRI features (centrally T2 hypointense lesion with peripheral edema and rim enhancement), the diagnosis of intradural and extramedullary spinal cord tuberculoma in the caseous stage was made.

The patient was started on a 4-drug anti-tuberculosis regimen (rifampicin, isoniazid, pyrazinamide and ethambutol). Following this treatment, his cough subsided, and notable improvement was seen in his neurologic function.

Spinal cord tuberculomas can present with various neurological symptoms, such as sensory and motor deficits, as well as bladder and bowel dysfunction, as observed in this case. The initial nonspecific symptoms including persistent cough and weight loss, likely delayed the diagnosis, leading to the development of the spinal lesion and neurological complications.

### *Case 2*

A 30-year-old male military personnel, stationed at a military camp for the past 2 years, presented with a 2-month history of productive cough and a 2-week history of lower back pain and numbness. Additionally, he had multiple matted and enlarged cervical lymph nodes.

No contact history with chronic cough was identified, and there was no family history of similar illnesses. The CBC showed mild lymphocytosis, the ESR was raised, and the chest X-ray revealed areas of patchy air space opacity.

AFB staining was performed on his sputum which tested positive 2 times, indicating the presence of Mycobacterium tuberculosis. Fine-needle aspiration cytology (FNAC) of the cervical lymph nodes confirmed caseous necrosis.

The spinal MRI revealed an oval, well-defined and intradural-intramedullary lesion measuring 14 × 12 mm located at the T12 vertebral level. On T1-weighted imaging, the lesion was isointense. On T2-weighted imaging, the lesion appeared hypointense and was surrounded by a hyperintense rim. On postgadolinium imaging, the lesion demonstrated peripheral rim enhancement, exhibiting the characteristic "target sign'' appearance which was in favor of central caseation. This was consistent with the caseous stage of CNS TB (Fig. 2).

Based on the clinical presentation, sputum AFB stain, FNAC findings, and the characteristic MRI features of T2-hypointensity with a hyperintense rim and ring enhancement, the results strongly suggested the caseous stage of spinal cord tuberculoma.

The patient was started on a standard anti-tubercular treatment regimen, including a combination of isoniazid, rifampicin, pyrazinamide, and ethambutol. The patient's symptoms gradually improved. Follow-up imaging at 6 months demonstrated significant reduction in the size of the tuberculoma (image not shown).

### *Case 3*

A 20-year-old female patient presented with a 3-month history of productive cough, a 1-month history of neck pain and

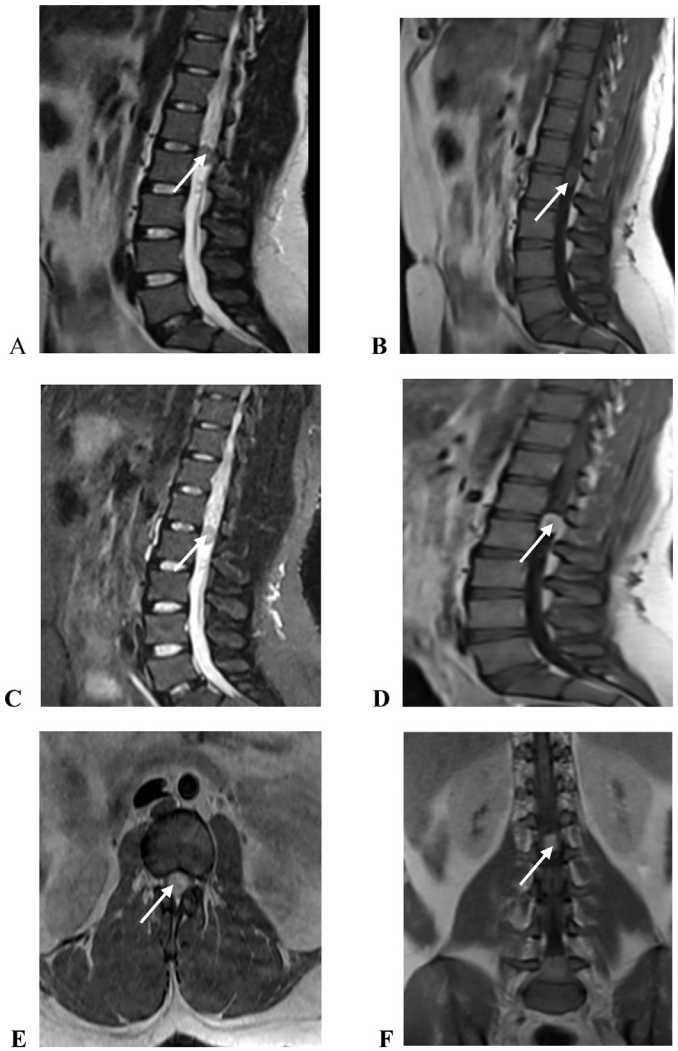



**Fig. 1 – Case 1. Twenty five-year-old male with spinal cord tuberculoma. The multiplanar spinal MRI (thoracolumbar) shows an intrathecal, extramedullary lesion measuring 16 × 12 mm at the L2 level. The lesion is isointense on T1W (arrow, B), iso to hypointense on T2W (arrow, A) and STIR (arrow, C) with a small focus of central hyperintensity on these sequences. On postgadolinium imaging, the lesion demonstrates primarily peripheral enhancement (arrows, D, E, F) which favors a differential of spinal TB caseation stage.**

deformity, and a 1-week history of quadriparesis. She also reported experiencing fever, unquantified weight loss, and night sweats.

No family history of similar illnesses or chronic cough was identified. The patient was given ceftriaxone antibiotics 1 month ago, resulting in partial improvement of signs and symptoms, which have since returned. The CBC is within the normal range, but the ESR is elevated. Acid Fast Bacilli (AFB) staining was performed on the patient's sputum, which tested positive on 2 occasions.

On multiplanar cervical spine MRI, there was an intradural-intramedullary well-defined lesion measuring 27 × 12 mm, located between the C2 and C4 vertebral levels in the cervical spine. The lesion displayed focal cord thickening, isointensity on T1 images and hypointensity on T2 images, accompanied by significant adjacent cord edema extending to brainstem. Postgadolinium imaging revealed peripheral enhancement, manifesting a target sign (Fig. 3). Additionally, there was no change in signal intensity in the adjacent vertebrae, and no intraspinal or paraspinal abscess collection was observed. The overlying musculature appeared normal.

Upon starting the 4-drug anti-TB regimen, signs and symptoms improved, appetite increased, and weight gain began.

This case illustrates cervical cord involvement, despite the thoracic cord being the most commonly affected area in spinal tuberculoma. The patient exhibited weakness in all extremities, highlighting the clinical significance of spinal tuberculoma in causing compressive myelopathy. The adjacent vertebral bone and paraspinal tissue appeared normal, indicating that the condition was not due to extension from surrounding tissues, but rather suggested possible hematogenous spread from a primary lung focus. Most spinal cord tuberculomas are associated with other central nervous system involvement, such as brain lesions or complications of pulmonary TB. In

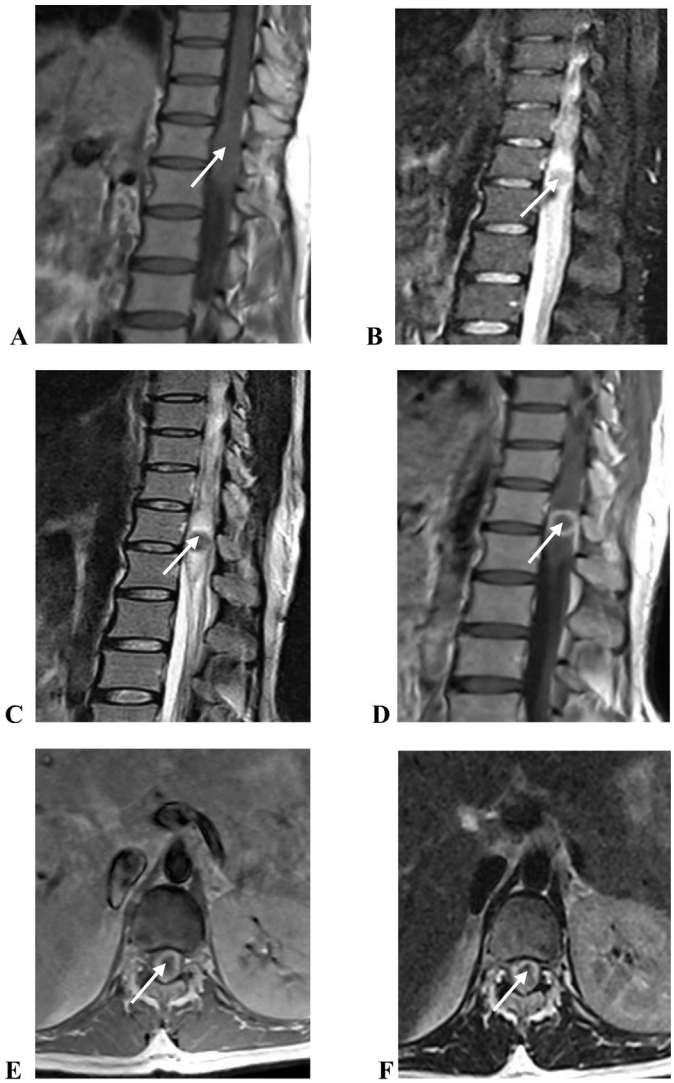



**Fig. 2 – Case 2. Thirty-year-old male with caseous stage spinal TB. The multiplanar, gadolinium-enhanced MRI shows an oval, well-defined and intradural-intramedullary lesion measuring 14 x 12 mm located at the T12 vertebral level within the spinal cord. On T1-weighted imaging, the lesion is isointense (arrow, A). On STIR and T2-weighted imaging, the lesion appears hypointense, surrounded by a hyperintense rim (arrows, B, C, D). On postgadolinium imaging, the lesion exhibits a peripheral smooth ring enhancement with central hypointensity, demonstrating the characteristic "target sign" appearance showing caseous stage spinal TB (arrows, E, F).**

this case, the patient presented with a chronic cough and a positive AFB sputum test, indicating hematogenous seeding from the primary lung focus. The typical intramedullary T2 hypointensity with peripheral ring enhancement strongly supported diagnosis at the caseous stage.

In all 3 cases, they were managed medically with anti-TB treatment, resulting in improved motor activity and cessation of symptoms, with no surgical intervention performed.

## Discussion

Isolated spinal cord tuberculomas, without the involvement of meninges and overlying bone, are extremely uncommon and have been reported only in case reports. This condition was first described by Abercrombie in 1828 [5], and since then, around 100 cases have been reported in the literature [5,6].

Spinal cord involvement in TB is much less common compared to intracranial tuberculomas, with only about 0.2% of CNS TB cases showing spinal cord involvement. As a result, it can be quite challenging to diagnose spinal TB, especially in individuals who are not from regions where the disease is endemic [7].

In contrast to Pott's disease, spinal cord TB lacks a significant adjacent inflammatory response, and the intervertebral disc is usually spared until the late stages of the disease. Spinal cord TB more frequently affects young adults, typically between the ages of 25 and 35, with the thoracic region of the spinal cord being the most commonly involved [8,9].

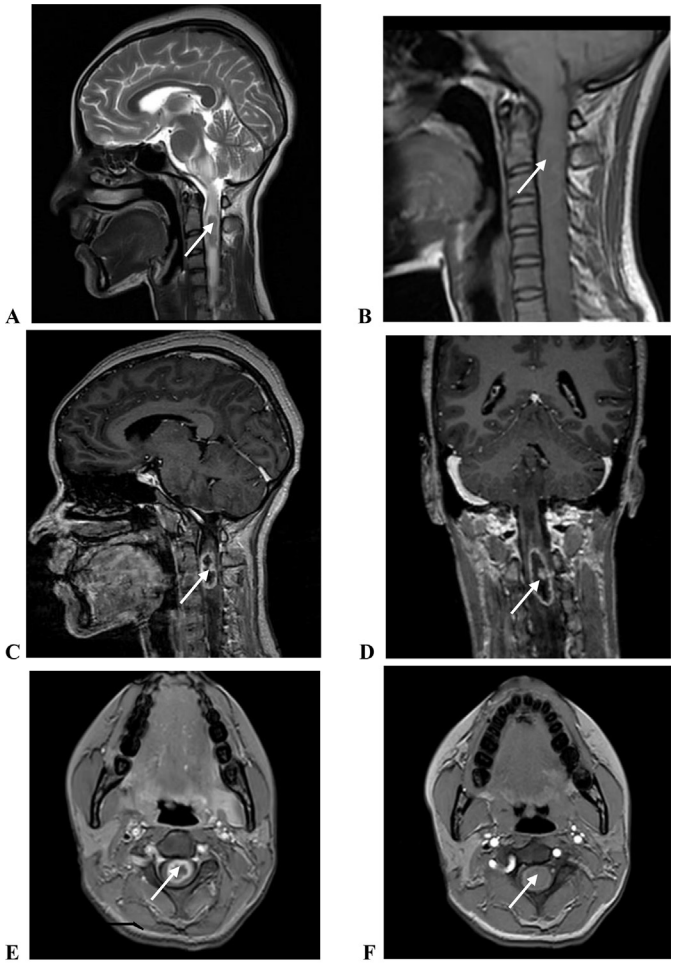



**Fig. 3 – Twenty-year-old female with caseous stage spinal TB. MRI findings of the brain and cervical spine reveal an intramedullary well defined mass lesion measuring 27 x 12 mm in the cervical cord between C2 and C4 vertebral levels. On the sagittal T1 images, the region appears iso-intense (arrows, A, F), while it is hypointense on T2 images (arrow, B). Postcontrast images show peripheral ring-like enhancement, indicated by the arrow (C, D, E). Additionally, the T2 images display marked adjacent hyperintensity extending up to the level of the medulla, along with adjacent edema.**

The spread of the infection to the spinal cord is usually hematogenous, although in some cases, spinal cord involvement may arise from local extension of a tuberculous focus in the vertebral spine itself. Prompt diagnosis and appropriate anti-tubercular treatment, along with potential surgical intervention if necessary, are crucial in managing spinal tuberculomas and preventing further neurological deterioration [7,9].

Spinal cord tuberculomas are often confused with spinal medullary lesions, such as spinal cord astrocytomas and ependymomas. Both types of lesions display T1-weighted hypointensity and T2-weighted hyperintensity; however, neither exhibits T2 hypointensity, which is characteristic of the caseous stage of TB, as seen in all our cases. These tumors can further confuse the diagnosis, particularly in the early noncaseous stage of tuberculoma, where T2 hyperintensity is seen [10–12]. To differentiate these, clinical associations, epidemiological factors, the extent and location of spinal cord involvement, and enhancement patterns can assist in making a correct diagnosis. Additionally, spinal cord abscesses represent another differential diagnosis, typically characterized by T2 hyperintensity with a hypointense rim. Nonetheless, T2 hypointensity remains a valuable tool for identification. In all the cases discussed, the presence of typical T2 hypointensity strongly suggests a diagnosis of tuberculoma in the caseous stage rather than other possible differentials [11,12].

A similar case report by Knobbe et al. describes a 71-year-old male who presented with extensive spinal involvement in the lower thoracic region, indicative of the caseous stage of TB [13]. The MRI findings were similar to those in ours, however, he was older and exhibited multi-level involvement from T7 to T12. Notably, he had a history of non-Hodgkin lymphoma and was undergoing chemotherapy, although he was HIV negative. In contrast, our patients are younger and demonstrate smaller segments of spinal cord involvement, likely due to the absence of any known immune-compromising conditions.

In 2 cases reported by Lu et al, the findings closely resemble those in our cases, as they both involved short segments of the spinal cord—one in the cervical region and the other in the lower thoracic region—exhibiting the typical target sign [14].

Overall, spinal cord TB is a relatively rare manifestation of this infectious disease, making it a diagnostic challenge, particularly for clinicians practicing outside tuberculosis-endemic areas [8].

Diagnosis of spinal TB is often suggested by characteristic clinical features and imaging findings, but tissue diagnosis is considered the gold standard. Bacteriological confirmation can be obtained through methods like culture, histopathology, or polymerase chain reaction (PCR) [15].

In the 3 cases presented, the patients' diagnoses and initiation of anti-TB treatment were based on their history, TB symptom complex, and physical exam findings of focal neurological deficits [16]. This diagnosis was further supported by suggestive MRI findings, revealing a characteristic "target sign'' lesion, as well as FNAC findings of necrotizing granulomatous inflammation from lymph nodes and positive AFB from sputum staining [17].

### *Magnetic resonance imaging (MRI)*

MRI is the preferred modality for the evaluation of spinal TB, with a reported sensitivity of 96% and specificity of 93%. The Infectious Diseases Society of America (IDSA) strongly recommends MRI in the evaluation of suspected spinal infections, as it can better assess spinal canal compromise and cord compression [14]. MRI signal changes can be observed as early as 3-5 days after spinal infection, when other imaging modalities may not show any abnormalities [18,19].

Contrast enhancement improves the diagnostic accuracy of spinal TB detection and helps evaluate the extent of disease, such as intraspinal canal extension or spinal cord involvement. Vertebral inflammatory changes are seen as marrow edema, appearing as hypointensity on T1-weighted images and hyperintensity on STIR and T2-weighted images [10,19].

With gadolinium, enhanced regions of marrow edema show well-defined heterogeneous enhancement. When there is paraspinal abscess, smooth, thin peripheral wall enhancement is seen. Disc involvement is typically seen later in the course of the disease and is characterized by reduced disc space, high signal intensity on T2-weighted images with loss of the intranuclear T2 hypointense cleft [10,18].

MRI can also demonstrate intramedullary or extramedullary tuberculomas, spinal cord edema, and myelomalacia. The radiological picture depends on the stage of the tuberculoma. MRI findings demonstrate 4 types of tuberculoma: noncaseating granuloma, caseating granuloma with a solid center, caseating granuloma with a liquid center, and calcified granuloma. In the early phase, tuberculoma is characterized by severe infective reactions, poor formation of the gelatinous capsule, and severe edema around the lesion. During this phase, T1-weighted and T2-weighted images both show equal signal intensity, which is evenly enhanced after contrast administration [16,18].

MRI findings for each stage are presented as follows.

1. Noncaseating granuloma
   In cases of noncaseating granuloma, MRI characteristics include iso- to hypointense signal on T1-weighted images and hyperintensity on T2-weighted images. FLAIR sequences show no suppression, and DWI and ADC maps demonstrate no restricted diffusion. After gadolinium contrast administration (T1 C+), there is typically homogeneous enhancement [6,12].
2. Caseating granuloma
   For caseating granulomas, T1-weighted images appear iso- to hypointense with a hyperintense rim. On T2-weighted images, central hypointensity with a hyperintense rim that represents gliosis and abundant monocyte infiltration is found, surrounded by vasogenic edema. FLAIR sequences also show no suppression, and DWI/ADC sequences exhibit no restricted diffusion. T1 C+ images may reveal either homogeneous or ring enhancement. Additionally, MR spectroscopy shows a decrease in N-acetylaspartate (NAA)/creatine (Cr) ratios, a slight decrease in NAA/choline (Cho) ratios, and elevated lipid-lactate peaks in about 86% of cases [6,10,12].
3. Caseating granuloma with central liquefaction
   In cases where caseating granulomas undergo central liquefaction, T1-weighted images still show iso- to hypointense signal with a hyperintense rim. T2-weighted images reveal a hypointense rim surrounding a central hyperintensity, accompanied by vasogenic edema. FLAIR imaging may exhibit partial suppression, while DWI/ADC results can show variable diffusion restriction. T1 C+ images typically demonstrate ring enhancement [6,10,12].
4. Calcified granuloma
   Calcified granulomas appear iso- to hypointense on T1-weighted images and hypointense on T2-weighted images. FLAIR sequences do not show suppression, and DWI/ADC images reveal no restriction. Following gadolinium administration (T1 C+), calcified granulomas generally show no enhancement [6,10,12].

As the gelatinous content in the tuberculoma increases, the peripheral edema is alleviated or may disappear. In this stage, T1-weighted images show equal signal intensity, while T2-weighted images show equal or low signal intensity, with rim enhancement and low signal in the central region which is the "target sign''. Low signal at the center represents caseous substance and the surrounding high signal rim shows peripheral infective granulation tissues. Details of characteristic MRI findings are more completely covered in the Introduction of Mohajeri Moghaddam and Bhatt [20].

### *Differential diagnosis*

Most of the differentials of spinal cord tuberculoma are spinal intramedullary tumors, such as astrocytic glioma, ependymoma, and hemangioblastoma or spinal cord abscess. The "target sign'' on MRI, as seen on two of the above cases, characterized by a central area of T2 low signal intensity surrounded by a peripheral rim of high signal intensity, is a valuable indicator that can help differentiate spinal tuberculoma from other intramedullary lesions. Rim enhancement on contrast-enhanced MRI is also commonly observed in spinal TB. Compared to tumors, spinal tuberculoma typically has a sharper margin and lower T2-weighted signal intensity, which can help in the differentiation of these entities [19].

## Conclusion

In these 3 cases of spinal TB, the MRI findings were specific, and an accurate diagnosis was obtained, which was supported by the clinical data. For the diagnosis of spinal intramedullary TB, MRI is the optimal imaging technique.

## Scope of the manuscript

The manuscript aims to consolidate the existing knowledge of imaging of spinal tuberculosis and the updates.

## Author contributions

All authors played a substantial role in conceptualizing, executing, and compiling the manuscript. The final version was approved by all authors for publication.

## Data availability statement

The datasets and images utilized in this manuscript will be made available by the corresponding author upon reasonable request.

## Patient consent

The patients involved have voluntarily consented to the publication of their cases, including any associated images.

REFERENCES

[1] Kubihal V, Sharma R, Krishna Kumar RG, Chandrashekhara SH. Imaging update in spinal tuberculosis. J Clin Orthop Trauma 2022;25:101742.
[2] Bagcchi S. WHO's global tuberculosis report 2022. Lancet Microbe 2023;4:e20.
[3] Rajasekaran S, Soundararajan DCR, Shetty AP, Kanna RM. Spinal tuberculosis: current concepts. Global Spine J 2018;8:96S–108S.
[4] Shanmuganathan R, Ramachandran K, Shetty AP, Kanna RM. Active tuberculosis of spine: current updates. N Am Spine Soc J 2023;16:100267.
[5] Abercrombie J. Pathological and practical researches on diseases of the brain and the spinal cord. Edinburgh: Waugh and Innes; 1828. p. 731–2.
[6] Arbelaez A, Medina E, Restrepo F. Cerebral tuberculosis. Semin Roentgenol 2004;39:474–81.
[7] Khanna K, Sabharwal S. Spinal tuberculosis: a comprehensive review for the modern spine surgeon. Spine J 2019;19:1858–70.
[8] Gupta M, Tobin EH, Munakomi S. CNS tuberculosis. Treasure Island, FL: StatPearls Publishing; 2024.
[9] Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK. Central nervous system tuberculosis: pathogenesis and clinical aspects. Clin Microbiol Rev 2008;21:243–61.
[10] Khatri GD, Krishnan V, Antil N, Saigal G. Magnetic resonance imaging spectrum of intracranial tubercular lesions: one disease, many faces. Pol J Radiol 2018;83:e524–35.
[11] Shih RY, Koeller KK. Intramedullary masses of the spinal cord: radiologic-pathologic correlation. Radiographics 2020;40:1125–45.
[12] Shukla R, Abbas A, Kumar P, Gupta RK, Jha S, Prasad KN. Evaluation of cerebral infarction in tuberculous meningitis by diffusion weighted imaging. J Infect 2008;57:298–306.
[13] Knobbe K, Gaines M. Intramedullary tuberculoma of the spinal cord. Kans J Med 2020;13:300–3.
[14] Lu M. Imaging diagnosis of spinal intramedullary tuberculoma: case reports and literature review. J Spinal Cord Med 2010;33:159–62.
[15] Rasouli MR, Mirkoohi M, Vaccaro AR, Yarandi KK, Rahimi-Movaghar V. Spinal tuberculosis: diagnosis and management. Asian Spine J 2012;6:294–308.
[16] Sanei Taheri M, Karimi MA, Haghighatkhah H, Pourghorban R, Samadian M, Kasmaei HD. Central nervous system tuberculosis: an imaging-focused review of a reemerging disease. Radiol Res Pract 2015;2015:202806.
[17] Huang Y, Ai L, Wang X, Wang F. Review and updates on the diagnosis of tuberculosis. J Clin Med 2022;11(19):5826.
[18] Lee KY. Comparison of pyogenic spondylitis and tuberculous spondylitis. Asian Spine J 2014;8:216–23.
[19] Nagashima H, Tanishima S, Tanida A. Diagnosis and management of spinal infections. J Orthop Sci 2018;23:8–13.
[20] Mohajeri Moghaddam S, Bhatt AA. Location, length, and enhancement: systematic approach to differentiating intramedullary spinal cord lesions. Insights Imaging 2018;9:511–26.